cise, nearly doubled his weight in less than twelve weeks. The movements of the legs were awkward, walking was difficult, and the left arm was completely paralyzed. There was such marked atrophy of the muscles as to suggest progressive muscular atrophy. In the third case, a coachman, there was tubercular neuritis with complete paralysis of the lower limbs, paresis in the arms, hoarseness due to paralysis of the vocal cords on the left side, and intense pains. Well-regulated diet caused all these symptoms to disappear, though the tubercular condition of the lungs remained the same. In the discussion which followed, Dr. Goldscheider remarked that implication of the cranial nerves was a rare thing in multiple neuritis; in one instance he had seen the motor oculi involved, and in another tachycardia, due to a lesion of the pneumogastric that was later followed by left-sided optic neuritis. Dr. Leyden then said that in his first work he had objected strongly to any dogmatic separation of peripheral nerves and the central system in their relations to multiple neuritis. A poisonous agent could simultaneously attack these two systems of nerves. According to his personal observation, anatomical lesions of the central system were found only in the ganglionic cells. There might also be disseminated lesions in the gray matter. Up to the present time he had found none in the white. Also, it seemed unwise to draw too deep lines of demarcation between nerve and muscle, for in this disease there might be primarily a myositis. The anatomical nerve lesions in multiple neuritis present some variety. There may be abundant proliferation of cells impinging upon nerve substance and upon the nerve sheath. Or degeneration of the nerve may occur and disappearance of myeline. This is the inflammatory form. Acute ascending paralysis should be classified as a multiple neuritis. The toxic agent first disturbs the function of the nerve, and this is followed by nerve degeneration. Whether multiple neuritis ever attacked the cord or not, Dr. Leyden was unable to state, never having encountered cases wherein tabes or myelitis had immediately followed this particular abnormity. Nevertheless, such a sequence was by no means an impossibility. L. F. B.

## CHARACTERISTICS OF HYSTERICAL TREMOR.

Dr. Odds has studied the general characteristics of this condition, of which the following summary appears in the Union Médicale, October 21, 1891.

*Cause.*—From this point of view there are three distinct forms of hysterical tremor. First, hystero-emotional (41 cases out of 55), due to sudden shock, fear, and slow depressing emotions, whether complicated or not by psychic depression. Then there is hysterotoxic tremor, similar to the tremor following infectious disease. And there is also tremor due to hysterical attacks, to apoplectiform or epileptoid seizures.

*Onset.*—This is often classic in its method. There is a prodromal period after shock, in which there is constant headache and intellectual or psychic abnormity.

*Quality of tremor when once established.*—Its variability is marked. It is spontaneous and induced by any hysterical accident, as emotion, a convulsion, etc. It is paradoxical on account of anomalies due to the tremor itself and secondary hysterical symptoms that accompany it. L. F. B.

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## CLINICAL.

# A CASE OF HEMIPLEGIA, FOLLOWED BY EARLY MUSCULAR ATROPHY.

In the "Neurologisches Centralblatt," No. 20, 1891, Dr. L. Darkchewitch reports a case of hemiplegia in which muscular atrophy appeared shortly after the paralysis. The patient was 43 years of age and without a history of alcoholism or syphilis. When first seen he was semi-conscious. Word-blindness and complete motor aphasia existed. There was also total right hemiplegia with involvement of the lower facial branches and the hypoglossal nerve. Sensation seemed to be deficient on the paralyzed side, and the tendon reflexes were somewhat exaggerated. Loss of control of bladder and rectum. The urine contained neither albumen nor sugar. Four weeks after the attack there was marked atrophy affecting the right deltoid, supra-spinatus, infra-spinatus and pectoralis major. The atrophy rapidly extended to the muscles of the forearm and the small muscles of the hand. The elecrical reactions were normal. About one month later death took place as a result of another cerebral lesion.

At the autopsy two spots of softening were found in the corpora striata, one on either side, thus accounting for the hemiplegia.

A careful microscopical examination was made of the brain, the spinal cord, the spinal nerve roots, the peripheral